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Commentary

Depression deconstruction lessons from psychosomatic research

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In the review by Poole and colleagues in this issue of *Journal of Psychosomatic Research*, it is suggested that depression that develops in the aftermath of an acute coronary syndrome (ACS), such as a myocardial infarction (MI), may have a different etiology than depression as is observed in the general population. Interestingly, the authors make a case that post-MI depression results from inflammatory processes that surround the MI [1].

The review fits well in the growing attention for the phenotypical heterogeneity of major depression which is observed both in general psychiatry and in the psychosomatic field (e.g., [2–5]). For instance, Lux and Kendler [2] recently evaluated the heterogeneity of depressive symptoms in a large sample of twins, distinguishing between cognitive and neurovegetative symptoms – a distinction that has found its way already in the post-MI depression literature (e.g., [4–6]). Interestingly, in this general population sample, they found that especially cognitive symptoms within the depression spectrum were associated with several clinical characteristics, including higher neuroticism and lower introversion scores, longer depression duration and more chronicity. In contrast, previous work on post-MI depression has suggested that specifically somatic or neurovegetative symptoms are associated with poor cardiovascular outcomes [6], probably via mechanisms linked to cardiovascular disease such as inflammation [7] but also heart rate variability [8].

To those working in psychosomatic medicine, it may not be surprising that attention to the heterogeneity of depression, in terms of both its presentation and etiology, has emerged predominantly in this field. Clinicians from consultation-liaison psychiatry and medical psychology are frequently confronted with medical patients presenting with feelings of distress, while often it remains unclear whether those feelings should be interpreted in the light of an adjustment disorder or be seen as symptoms of major depressive disorder (MDD), a somatic reaction to a physiological process or an adaptive psychological reaction to a life-threatening stressor. Unfortunately, the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*, has not been particularly helpful in clarifying this issue by excluding symptoms as part of depression only when they are the

'necessary consequence of drugs or a physical condition' [9]. In a similar vein, screening instruments for depression were once designed for use in the general population and evaluated as such [10], while in medical patients, such instruments are known to be notoriously overinclusive. This may well be due to their failure to adequately address the etiological basis of symptoms during a quick self-report screen [11]. Yet, especially because of the complex etiology of depression-like symptoms in medical patients, the field of psychosomatic medicine may play a pioneering role in deconstructing depression into clinically relevant subtypes.

The conclusion drawn by Poole and colleagues that inflammation-based depression may be an important subtype of depression that is specifically prevalent in post-MI populations builds on a large and heterogeneous body of literature. The authors have performed an impressive job, as they integrated quite varying parts of the literature, including animal research, epidemiological studies and intervention trials, and should be complimented for that. Still, several issues remain, particularly with respect to how inflammation-based depression may best be dealt with. One of the pillars on which Poole and colleagues build their argument is that clinical trials evaluating antidepressant treatment in ACS patients have not been very successful. Indeed, as only 1%–4% of the improvement in depressive symptoms may be attributed to the specific ingredients of antidepressants, we are still far away from a truly effective treatment. Yet, these figures are not necessarily worse than for unselected patients (mostly without ACS). For instance, in *cardiac patients*, the treatment effects on depressive symptoms after 8 weeks for four antidepressants (fluoxetine, sertraline, mirtazapine and citalopram) were highly similar to those reported by Turner and colleagues in *unselected patients* using FDA data [11,12]. Similarly, although randomised comparisons of psychotherapeutic interventions have not always been very successful, there is not much reason to believe that these interventions are less effective in patients with ACS than in those without.

One interesting aspect that Poole and colleagues touched upon is the reference to the fascinating work by Appels and colleagues on vital exhaustion. The concept of vital exhaustion was developed first as a form of mental and physical distress preceding ACS, but later on also as a sequel of ACS with potentially cardiotoxic properties [13]. Of interest, vital exhaustion was thought to consist of a combination of feelings of exhaustion and irritation, while depressed mood and negative cognitions are not frequently present in this syndrome. Its association with and delineation from depression has received quite some attention, and the question of whether it represents a form of distress different from depression has been a pertinent one [14]. Poole and colleagues' concept of an inflammation-based somatic subtype of

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depression is slightly different than vital exhaustion as they consider it to be a subtype of depression, i.e., fulfilling the criteria for MDD. Still, perhaps insights from the literature on the etiology (e.g., the role of inflammation in vital exhaustion [15]) and treatment of vital exhaustion (e.g., relaxation therapy, [16]) might help in improving the efficacy of depression treatment.

I believe that Poole and colleagues are teaching us a valuable lesson in depression deconstruction, and this may well be a lesson that goes beyond the field of cardiopsychiatry and psychosomatic research. Only the future can tell whether the identification of inflammation-based depression will be truly influential. Its relevance, I believe, will depend on the question of whether we will be able to develop and test interventions that are specifically effective in reducing inflammation-based symptoms of depression.

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